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Influence of Passive Smoking on Pulmonary Function: A Survey[1,2]

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A review of the effects of passive smoking on pulmonary function has been made. In children, there is still a great deal of confusion and controversy. The range of the many effects studied is limited; most prospective studies show small effects. In general, it appears that respiratory infections in children may be increased with passive smoking. The effects on children's pulmonary function ranges from 0 to 3% over the age range from conception to 20 years; these differences in absolute magnitude are considered small. In healthy adults, the effects on pulmonary function and symptoms are not considered to be of concern; the effects in asthmatics require further study. A study to evaluate the above effects is presented here. It derives from a prospective study of airway obstructive diseases in Tucson, Arizona. From that study, it was concluded that the effect of passive smoking on pulmonary function or respiratory symptoms (recorded on a daily basis) in children is not positive. Effects may be seen with regard to responses to other irritants in passive-smoking children. In adults, no effect was seen, even in those with asthma or airway obstructive diseases; this may be due to low dosage. It is concluded that further, more appropriate studies are needed to understand the role of passive smoking on pulmonary function. © 1984 Academic Press, Inc.

INTRODUCTION

Although there is substantial agreement that passive, or involuntary, tobacco smoking produces annoyance and sensory irritant effects, its role in producing cardiovascular or respiratory diseases is less clear (34, 47). The specific contributions of passive smoke to personal exposures and their effects have not been sufficiently documented.

Several studies have shown that the levels of carbon monoxide and other sidestream smoke constituents in indoor areas are high in many instances (34, 43, 46, 47). Deposition of sidestream cigarette smoke in the human respiratory tract has been shown to be only about 11%, much less than mainstream smoke (16). The impact of environmental tobacco on indoor concentrations of nitrogen dioxide (NO_2) is usually small compared with the impact of gas stoves (64). Blood carbon monoxide (CO) levels may be higher in those exposed to passive smoke (36, 42), but are probably lower than the carboxyhemoglobin (COHb) levels associated with exposures to unvented gas stoves (34). Some of the same respiratory diseases may occur in indoor situations in which high levels of respirable suspended particulates (RSP) and gases are due to wood-burning or kerosene heaters (1, 53) and not to environmental tobacco smoke.

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PULMONARY FUNCTION IN ADULTS

There have been several studies of passive smoking and respiratory impairment in adults. Comstock *et al.* (6) evaluated respiratory symptoms and lung function in a community population and found some effects from gas stoves but no effects from passive smoking; no exposure measurements were made.

Lebowitz and his co-workers (21, 22) studied chronic respiratory symptoms and lung function in a community population and found no effects of passive smoking.

Kauffmann *et al.* (18) studied pulmonary function in adult populations in France (without indoor measurements or information about use of gas appliances). They found some effects of spouse's smoking on nonsmoking women, but the healthiest population lived in the most polluted areas. This indicated that the study might have been flawed by biased population selection, biased testing, or confounding factors. One epidemiological study of chronic bronchitis in Czechoslovakia (52) showed a significant increase of symptoms of this disease in passive smokers, but this occurred almost entirely among men and showed a much greater relation to other adverse factors and/or exposures.

The Harvard Six City Study (59) performed covariance analyses for adults, examining passive smoking, gas stove usage, and ambient pollution (by city) and controlling for age, sex, and smoking. It found no effect of passive smoking on the adult respiratory system. Other population studies reported at a 1983 NIH workshop (33) led to the conclusion that there was basically no effect on pulmonary function and maybe only slight, inconsistent effects on symptoms in adults.

White and Froeb (60) studied self-selected volunteers with regard to workplace exposure and reported some effects of passive smoking in a subset of their volunteers; their study has been discussed in detail (23). Even with a biased population, poor study design, and incorrect statistical evaluation, there were no clear-cut, consistent, medically meaningful differences between passive smokers and groups of nonsmokers; a corrected statistical analysis strengthened this conclusion.

Three physiological studies of the effects of environmental tobacco smoke exposure on healthy subjects have been conducted, and all have been negative with regard to such effects (39, 51), even when massive amounts of smoke were used to expose nonsmokers (41). Two similar studies of the effects of environmental smoking on asthmatics and controls have demonstrated conflicting findings. One study (50) showed no important effect on asthmatics or controls, whereas the other study (7) demonstrated a broncho-constrictor effect in highly reactive asthmatics at higher concentrations.

PULMONARY FUNCTION IN CHILDREN

Studies of the relationship of children's lung function to parental smoking habits again show somewhat contradictory evidence. In some studies, such as those of Martinez *et al.* (29), Yarnell and St. Leger (63), and Kauffmann and Brille (17), effects of parental smoking were seen in terms of children's lung function; however, these did not measure or control for other indoor (or outdoor) pollutants.

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In other studies in which measurements were not made, contradictory results were found. Hasselblad *et al.* (15) demonstrated that maternal smoking habits explain 0.1% of the variance in children's forced expiratory volume (FEV) in the first 0.75 sec of the expiratory maneuver ($FEV_{0.75}$), but they utilized data from previous studies that had shown no such trend when examining other risk factors at the time (4, 14). Gas stove usage was not found to be significant in either study. Ekwo *et al.* (10) found respiratory effects on children from gas stove usage but inconsistent effects of passive smoking. Tager *et al.* (57) and Schenker *et al.* (48) found effects of parental smoking but not gas stoves; social status factors were again shown to be important.

Recently, several studies of passive smoking effects that included environmental monitoring have been performed. The results have been quite contradictory. In the studies by Florey *et al.* and Melia (11, 30, 31), looking at other indoor pollutants, they could find no relation of passive smoking to children's lung functions. Speizer *et al.* (55, 56) demonstrated the effects of gas stoves but not of passive smoking on children. In the same study, the reverse was shown with different analyses of further data (59), but there were inconsistencies between different cohorts, even in the same city. Dodge (8, 9) examined children's lung function cross-sectionally and logitudinally; he did not find any effect of gas stove usage or passive smoking, although self-reported cough was higher in children exposed to parental smoking. Studies by Binder *et al.* and Schilling *et al.* (2, 49) found indoor levels of RSP higher in Connecticut homes with smokers, but no respiratory effects were seen in adults or children. Often, results have been inconsistent within the same study, or a single positive finding has occurred without consideration for the number of comparisons examined.

The 1983 Geneva Workshop on Environmental Smoke (47) concluded that no definitive studies of acute effects in children have been performed. Furthermore, there was difficulty in interpreting several of the studies due to the large numbers of children involved and the qualitative responses found. Additionally the specific mechanisms of some irritant gases [such as sulfur dioxide (SO_2), ozone (O_3), and formaldehyde] involved are also unknown.

Retrospective studies of the etiology of respiratory diseases, such as asthma (13), have been discounted because of the major problem of reporting bias (4, 21). Patients with allergies appear to complain more of negative responses to environmental tobacco smoke (35, 40, 45, 54), as do many nonsmokers, but subjective responses generally have not been validated by epidemiological studies. The question also has been raised as to whether levels of environmental smoke per se found in the workplace could be considered sufficient to produce lung function effects in workers (58). There has been little evidence that passive smoking has an effect on chronic symptoms in adults (33, 47).

In Tucson, we have attempted to investigate the relationship between peak flow and reported respiratory symptoms and passive smoking, air pollutants, and aeroallergens (pollen, bacilli, fungi, algae) in the microenvironment of the home as well as the macro- (ambient) environment.

METHODS

A total of 117 families (229 adult and child subjects) in Tucson were randomly

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chosen in a stratified sample of families in uniform geographic clusters from a representative community population [already under study (20)], as previously described (23, 26). Studies of the total population sample indicated the presence of relationships between weekly symptoms and both air pollutants and allergens, controlling for weather (44). Stratification ensured that the families were representative of families with and without asthma in their geographic clusters. They were studied over a 2-year period, using daily symptom diaries and peak flow meters; daily response rates and peak flows were acceptable for a majority of days in all seasons (23, 28). All families provided environmental information as to characteristics of the house, yard, and streets, heating, cooling, type of stove, water heater, washer and dryer, and smoking in the home (26).

Daily ambient monitoring data for air pollutants, aero-allergens, and meteorological variables with regional and geographic clusters were used (23). Indoor and yard (micro-outdoor) monitorings were conducted in a random cluster sample representative of all study households (23, 26) for particulate matter [RSP and total suspended particulate matter (TSP)], pollen, bacilli, fungi, and algae, for 72 hr each, and grab samples were made for CO and O_3. Microenvironmental sampling of pollen, algae, and fungi have been described previously (23, 37, 38). Temperature and humidity varied by season, but the former did not vary very much indoors over the year and the latter did not differ much between indoors and outdoors over the year.

TSP indoors ranged from 5.7 to 68.5 $\mu g/m^3$, and simultaneous micro-outdoor TSP ranged from 2.1 to 169.6 $\mu g/m^3$. Cyclone measurements of RSP had a maximum of 49.8 $\mu g/m^3$ indoors and 124.5 $\mu g/m^3$ outdoors. Electron microscopy yielded little identifying information on the type of dust found on the floors of homes. Infiltration of outdoor TSP indoors was similar to pollen infiltration: total micro-indoor pollen counts were approximately 5% of the micro-outdoor pollen counts, which were about 5% of macro-pollen rates. Both TSP and RSP were significantly associated with environmental tobacco smoke (Table 1), but not with gas stove usage (26).

Averages of the two spot CO readings using infrared determinations were lower than 2.4 ppm; micro-outdoor readings were less than 3.8 ppm. Indoor CO was significantly associated with gas stove usage (26), but not with environmental tobacco smoke (Table 1), nor with gas-burning furnaces, water heaters, or washer–dryers (that is, vented appliances). Although low, it was used as the indicator pollutant measure of combustion emission from the gas stoves. Neither environmental tobacco smoke nor gas stove usage was associated with social status in this sample.

RESULTS

We found many interactions between indoor and indoor–outdoor pollutants, and weather, with respect to health effects. Children's standardized peak flow (PEF) was related to outdoor environmental factors only (O_3, TSP, temperature, and relative humidity); outdoor factors affected nonsmokers more than passive smokers (Table 2).

Adult PEF was related to active smoking (as were chronic symptoms), gas

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TABLE 1
TOTAL SUSPENDED PARTICULATES (TSP), RESPIRABLE SUSPENDED PARTICULATES (RSP), AND CARBON MONOXIDE (CO) BY ENVIRONMENTAL TOBACCO SMOKE

	ETS	
	No	Yes
TSP ($\mu g/m^3$)		
Mean	17.7	33.0*
SD	10.7	18.2
N	14	19
RSP ($\mu g/m^3$)		
Mean	7.2	13.1**
SD	8.0	13.1
N	15	19
CO ($\mu g/m^3$)		
Mean	1.2	1.2
SD	0.6	0.6
N	14	12

* Difference by two-tailed *t* test, $P < 0.005$.
** Borderline significant (two-tailed *t* test) $0.05 < P < 0.10$.

stove use, and outdoor NO_2 (independent and interactive) in asthmatics, allergics, and normals. In some cases, PEF was related to outdoor O_3, temperature, and relative humidity in nonnormal groups combined.

PEF in adult asthmatics was related also to temperature, high relative humidity, micro-pollen, and indoor TSP (Table 3), but not to passive smoking. "Asthma attacks" in adult asthmatics were related to temperature and to indoor TSP and RSP, but not to passive smoking or outdoor TSP (28).

Indoor TSP (but not passive smoking) was associated with rhinitis in asthmatics and nonasthmatics, as was RSP. Eye irritation in all adults and increased productive cough in nonasthmatics also were related to indoor TSP, but not to passive smoking. In addition, gas stove usage (and indoor CO) was associated with increased rhinitis in all adults, increased productive cough in asthmatics and allergics, increased sore throat in nonasthmatics, and eye irritation in all adults. Micro-pollen was associated also with adult eye irritation and rhinitis (in summer).

TABLE 2
CHILDREN'S MEAN PEAK FLOWS (EXPRESSED AS STANDARDIZED DEVIATION SCORES[a]: EFFECT OF OUTSIDE OZONE IN PASSIVE AND NONSMOKERS (PERSON DAYS = 1,566)

	Outside ozone			
	Very low	Low	Moderate	High[b]
Nonsmokers[c]	0.075	0.059	−0.175	−0.484[d]
Passive smokers	0.071	−0.041	−0.046	−0.079

Note. Overall ANOVA $P < 0.0001$.
[a] Deviations of individual values from seasonal means, standardized for seasonal variation.
[b] High: Max hourly O_3 (ppm) > 0.08.
[c] Analysis of variance, $P < 0.001$.
[d] $\Delta(\%) = -18 \pm \%$, significantly greater day-to-day change than seen in normals (22).

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TABLE 3
ASSOCIATIONS OF INDOOR TSP WITH AVERAGE PEAK FLOWS (STANDARDIZED DEVIATION SCORES) IN ADULT ASTHMATICS

PEF	Indoor TSP		
	Low	Moderate[a]	All
Mean	−1.17	−1.92	−1.56
SD	0.99	1.83	1.54
Person days	1,205	1,334	2,539

Note. Analysis of variance, $P < 0.0001$. Deviations of individual values from seasonal means, standardized for seasonal variation.
[a] 25+ μg/m³.

Thus, sensitive groups (children, asthmatics, allergics, AOD subjects) had more reactions to indoor and outdoor pollutants. Children were found to spend more time outdoors, usually with exercise, and were affected by outdoor pollutants more; these results confirm previous findings in Tucson (19). Indoor pollutants affected sensitive adults more than did outdoor pollutants. Indoor TSP was associated with health effects, but not passive smoking per se, indicating that other characteristics of indoor TSP were important (28).

We have shown that active smokers have higher circulating immunoglobulin E (IgE), which with smoking is related to airway obstructive diseases (3). However, we were not able to see any increase in IgE in passive-smoking children (Table 4). Generally, our recent findings are compatible with our previous negative findings (21, 22, 33). However, the blunting of irritant effects in children (seen in Table 2) needs further explanation.

DISCUSSION

Other effects of passive smoking have been reviewed recently (27, 47). The studies performed so far continue to provide confusing results vis-à-vis most public health issues. Many of the problems of positive studies are due to poor study design related to reporting bias, lack of control for family concordance, interaction among various pollutant emitters, lack of appropriate measurements (61), and crude statistical studies of geographical differences.

Confounding factors need to be incorporated more explicitly and completely.

TABLE 4
CIRCULATING LOG IMMUNOGLOBULIN E (IgE) IN CHILDREN BY CURRENT PARENTAL SMOKING

Parental smoking	N	Log IgE	
		Mean	SD
Both smoke	58	1.76	0.77
Father smokes	86	1.85	0.69
Mother smokes	28	2.09	0.64
Neither smoke	184	1.91	0.80

Note. Analysis of variance, $P = 0.258$.

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Better ascertainment and control of the covariables and confounders are needed (12). These include the following:

(a) Unvented combustion products from combustion appliances: gas (especially stoves), wood burning, and kerosene burning.

(b) Effects of other indoor pollutants (e.g., formaldehyde, consumer products), mostly irritant in nature; including interaction effects.

(c) Other indoor conditions of biological importance, such as (i) temperature and humidity conditions, size of house, ventilation (e.g., air exchange rates); (ii) pollen, molds, mites, and other allergens; and (iii) infectious organisms.

(d) Outdoor and occupational exposures and personal mobility, representing total/integrated exposure (includes consideration of geographic region, urban/rural setting).

(e) Socioeconomic status, cultural, ethnic, and religious factors (re: crowding, number of siblings, household conditions, occupation of parents, host conditions, risk of infectious spread, medical knowledge, medical care, reporting biases, etc.).

(f) Demographic and medical characteristics of the study population, such as (i) age, sex, marital status; (ii) presence of underlying respiratory condition (e.g., bronchial reactivity, clinical airways obstruction, atopy); (iii) disability and/or comorbidity.

(g) Parental syndromes, like productive cough, which would mask other processes/mechanisms (e.g., genetic, infectious) or affect reporting (4, 21) (e.g., winter morning phlegm).

(h) Maternal smoking during pregnancy and resulting effects on fetus and newborn, including low birth weight (33, 46).

(i) Annoyance responses, including odor problems, other psychological or social responses to tobacco smoking in a nonsmoker.

(j) Unknown smoking by subject.

A major problem to be resolved is the determination of the actual dose to the passive smoker (34, 61, 62). Monitoring of airborne particulates and organics is needed (47). Respirable particulates indoors are usually driven by smoking (2, 26, 64). However, the smoking-generated fraction of these concentrations is not related to health effects (28, 49). This implies that sizing and speciation of particles are necessary (61, 62). The differentiation of sources and concentrations would be very useful in determining mechanism of effects as well as the sources of primary concern.

Friedman and colleagues (12) showed, by questionnaire responses of about 38,000 adults, that 63% of people were exposed to environmental tobacco smoke, but their reported exposure was poorly correlated with serum thiocyanate levels. Exposures were correlated significantly with other health-related characteristics, including age, use of alcohol and marijuana, and exposure to occupational hazards. They were also correlated with sex, race, lower educational status, and single marital status. The reported concordance of marital smoking habits by categorization of spouse's smoking resulted in considerable misclassification, indicating spouse's smoking habits are a very inaccurate index of exposure. Future studies of passive-smoking effects should thus take these findings into account.

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The WHO/EURO Working Group (62) examined passive smoking within the context of all indoor exposures. It was felt that the current levels of knowledge did not yet allow quantitative assessment of the impact on the public health, as the knowledge of exposure effect relationships was inadequate, especially for delayed effects of chronic exposures. In terms of exposure levels, there were no data on how many cigarettes were being smoked in each of those indoor places where passive smoking can occur, and the ability to estimate how this is distributed within the building stock is marginal. It was felt that the amount of current information about the fraction of the population exposed to levels high enough to be a public health concern is smaller. The amount of information about the specific pollutants that might be responsible for the effects of concern is also very small. Of the variety of potential adverse effects, it was felt that only for irritation, odor, and airway response is there even a marginal amount of current knowledge. This group also felt that there were many possible pollutant interactions and that there were many other confounding exposures involved in examining the effects of passive smoking. Thus, in terms of relative priority, effects of passive smoking were low in comparison with other concerns, based on knowledge levels. Since, however, passive smoking may indeed be an important public health concern, it should be studied further. Attempts are currently being made by various groups to consider potential efficacious methods for further studies of the health effects of passive smoking (33, 47).

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